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SOCIAL COGNITION IN THE DIFFERENTIAL DIAGNOSIS OF AUTISM SPECTRUM DISORDERS AND PERSONALITY DISORDERS

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Abstract

Average intelligent patients with autism spectrum disorders (ASD) and patients with personality disorders (PD) are expected to show different problems in social cognition. Consequently, measuring social cognition may contribute to a better understanding and differentiation of ASD and PD. Therefore, we explored social cognition in these patient groups. Tests included the Mayer-Salovey-Caruso-Emotional-Intelligence-Test (MSCEIT) and Emotional-Quotient-Inventory (EQ-i). Analyses indicate that the ASD patients estimate themselves as more impaired on the ability to read emotions, but better on intrapersonal functioning, than the PD patients. In addition, both patient groups show more social cognitive impairment as compared to age and sex matched non-patient data. This holds true both for the self-report measure and part of an ability measure. Further research involving a dimensional approach and detailed profiling of strengths and weaknesses is advised to gain better understanding of the specificity and intensity of impairments, and of further differences between these disorders.

Key words: neurocognitive functions, emotional intelligence, Theory of Mind, autism spectrum disorders, personality disorders, social cognition, emotion

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Autism spectrum disorders (ASD) and Personality disorders (PD) are psychiatric diagnoses that can be difficult to differentiate in clinical practice, particularly when observing overt behaviour in patients with average (or higher) intelligence quotients (Fitzgerald 1999, Horwitz et al. 2004, Lehnhardt et al. 2013, Lugnegard et al. 2011, Kumbier et al. 2009). Lugnegard et al. (2011) studied 54 adults with an Autism spectrum disorder, and half of them also reached criteria for a personality disorder. Both patients with ASD and patients with PD show pervasive problems in interpersonal -social-behaviour and affective areas (e.g.; Egger et al. 2005, Esterberg et al. 2008, Frith 2003, Horwitz et al. 2004, Hurst et al. 2007, Lugnegard et al. 2011, Widiger et al. 2009). Horwitz et al. (2004) focussed on the phenomenological overlap between ASD and PD and mentioned social withdrawal behaviour as a resemblance between ASD and schizoid/schizotypal PD, impairments in empathic abilities as a resemblance

between ASD and antisocial and narcissistic PD and problems in social interaction and inflexibility as a resemblance between ASD and respectively avoidant and obsessive compulsive PD. Lehnhardt et al. more recently also focused on these overlapping problems (2013), which complicate differential diagnosis and offer the challenge for the present study.

The leading diagnostic classification systems, based on primarily behavioural symptoms, do not sufficiently contribute to this differential diagnostics dilemma; these systems rather confirm the behavioural overlap in symptoms instead of shedding light on underlying impairments. It accentuates the mentioned clinical practice dilemma of differential diagnostics for disorders like ASD and PD, which becomes markedly visible when it concerns patient groups with an average intelligence quotient. The variety in patients with ASD, and the variety in patients with PD make it heterogeneous patient groups. In order to be able to compare these

groups, we matched for intelligence and age, and sex was a covariate. For ASD, typical diagnostic practice involves amnestic and hetero-anamnestic information concerning one's (problem) behaviour in past and present, for which an interview based on ASD criteria can be used and neurocognitive (e.g. social emotional) test measures can be taken along with self-report questionnaires concerning ASD criteria. However, to date, there is no ideal test-protocol (Nederlandse Vereniging voor Psychiatrie/Nederlands Instituut van Psychologen 2013). Most of the patients involved in the present study and diagnosed with ASD got their classification after several of these diagnostic steps were taken. For PD, common diagnostic practice involves amnestic information concerning one's present problem behaviour, for which a questionnaire and interview based on DSM-IV criteria (e.g. Structured Clinical Interview for DSM-IV Disorders) can be used (Landelijke Stuurgroep voor Multidisciplinaire Richtlijnontwikkeling in de GGZ 2008, Weertman et al. 2000).

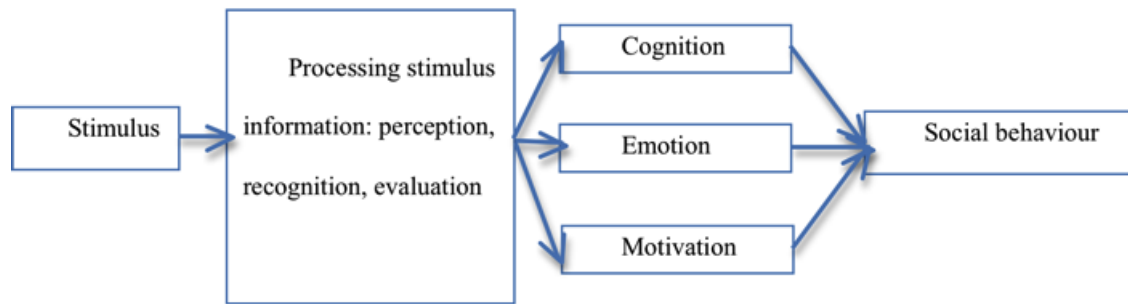
The brain-behaviour model illustrates how neurobiology (genes, brain) pairs with overt behaviour via neurocognitive functions like attention, social cognition, memory, language and planning. Neurocognitive functions typically concern the ability to process information (perception, recognition, evaluation of stimuli), and direct behaviour (Kandel et al. 2013). They form one of the measurable links between surface behaviour and the brain. Although neurocognitive function as measured in the present study does not involve bio-physiological measures like EEG/MRI, it does get under the surface of overt behaviour. Evolving from this, knowledge of neurocognitive functioning in disorders like ASD and PD is thought to lead to a better understanding of the pervasive (behavioural) problems in, and differentiation between these disorders, thereby dealing with the earlier mentioned clinical practice dilemma. The overt behavioural perspective involves observations that one is not making eye contact, whereas the "neurocognitive" perspective takes a check under the surface, looking for the key issues that impair the eye contact (for example, shyness, an inability to read minds or signs of visual impairment). Both perspectives are essentially useful, but when one wants to achieve adequate social behavioural change (like improvement of eye contact), the neurocognitive check under the surface is needed to identify the key impairments on which indications for treatment can be based (e.g. glasses, training of facial emotion recognition and/or therapeutically improving one's self-confidence and assertiveness in social interactions). Looking back at the clinical practice dilemma, especially when an average intelligent person with ASD and a person with PD show primary problems in social interaction, whereas other problems are thought to be less prominently present (e.g. problems in communication and impulse control), the differentiation between the disorders can be most difficult. The primary neurocognitive function that underlies interpersonal functioning is social cognition.

Social cognition broadly represents the integrated processing of socio-emotional information, needed to direct interaction, thereby underlying adaptive social behaviour (Swaab et al. 2011). It involves the perception of others (e.g. body signs, facial expressions, intentions), the perception of self (e.g. own thoughts and emotions) and interpersonal knowledge needed to manage tasks and self-regulate daily life. Thoughts and emotion play an influencing role in both the processing of the incoming information and in the behavioural output (Adolphs 2001, 2006 and 2009, Beer and Ochsner 2006, Swaab et al. 2011). For instance, the

presence of a (cognitive) negativity bias and/or sad emotion influences the way in which contextual (social) information is perceived (primarily giving attention to negative contextual information instead of to neutral or positive information), and this in turn influences and regulates one's planning, decision-making and behavioural actions as well (Izard 2009, Vaish et al. 2008). Social cognition is involved in human empathy (the experiential understanding of other's intentions and emotions) and is fundamentally needed to control affect, drive and motivation. Elaborating on all of this, social cognition forms the basis to come to adjusted (social) behaviour (Decety 2010, Johnson et al. 2003). This makes social cognition a relevant research topic, when having differential diagnostics dilemmas concerning deviant social behaviour.

Social cognition has earlier been a focus of studies concerning various neurodegenerative disorders and schizophrenia. Social withdrawal behaviour, problems in emotion recognition and Theory of Mind impairments (the ability to attribute intentions, thoughts and/or emotions to others, and thereby understanding situations from perspectives other than one's own (Premack and Woodruff 1978) are profound characteristics of ASD (e.g. APA 1994, Bonshtein et al. 2006, Couture et al. 2010, Eack et al. 2010, Edwards et al. 2002, Lough and Hodges 2002, Piskulic et al. 2010). Furthermore, impairments in empathy, face processing, social attribution, affective contact and affect regulation are common in ASD (e.g., Atkinson 2009, Baron-Cohen et al. 1997, Baron-Cohen and Wheelwright 2004, Domes et al. 2008, Grossman et al. 2000, review of Hill and Frith 2003, Kanner 1943, Konstantareas and Steward 2006). Whereas disorders like schizophrenia and ASD have been compared (Pinkham et al. 2008), studies performed on PD do not have a rich history of searching for correlations with neuropsychiatric disorders like ASD (Horwitz et al. 2004, Lehnhardt et al. 2013). However, the clinically experienced difficulties to differentiate ASD and PD offer a relevant challenge to test the justification of this lack of research. The history of studies involving social cognition and PD shows a tendency to link Borderline PD with social cognitive problems like disturbed recognition of emotions, thoughts and intentions in others (e.g., Fonagy et al. 2003, Kernberg 1996, Preißler et al. 2010, Westen 1991). The anti-social and schizotypal PD were associated with facial emotion recognition impairments (e.g., Brown and Cohen 2010, Marsh and Blair 2007). The ability for Theory of Mind was, as far as studied, not impaired for patients with Borderline PD and for patients with a more Avoidant/withdrawing PD (Arntz et al. 2009, Franzen et al. 2011). Patients with Borderline PD do tend to show a negativity/anger bias, and a heightened sensitivity to the detection of (particular facial expressions of) negative emotions (Domes et al. 2009, Lynch et al. 2006). With respect to the regulative side of the social cognitive process, Borderline PD has been linked to impairments in emotion regulation (e.g., Dijke et al. 2010, Johnson et al. 2003). Blair (2008) combined ASD and psychopathy because of overlapping empathic impairments in his study, and linked psychopathy to impairments in emotional empathy and autism to problems in cognitive empathy (Theory of Mind).

As described above, social cognitive impairments have been shown in both PD and ASD; problems in PD seem to be linked to the emotionalising part of the social cognitive process, whereas for ASD, both the cognitive and emotionalising part of the process are shown to be impaired. However, to our best knowledge,

Figure 1. *Social cognitive model (Adolphs 2001)*

groups of patients with ASD and PD have hardly ever been compared in one study involving the same tasks and conditions.

The aim of our present study is to contribute to the understanding and differentiation of ASD and PD, by mapping several aspects of social cognition, thereby studying parts of the phenomenological overlap between ASD and PD that complicate differential diagnostics in clinical practice. Studying the described neurocognitive chain of perceptive, emotional and executive processes that eventually result in social behaviour will give relevant information about the nature and intensity of the core problems in ASD and PD. It can thereby contribute to clarification of the mentioned differential diagnostics dilemma. Furthermore, we expect that this neurocognitive perspective on pathology can contribute to tailored psychological assessment and basic treatment indications to promote adjusted (self-regulative) daily life participation, by focusing on strengths and weaknesses in the functional drive of behaviour rather than solely on overt observations.

The main question in this study is: do patients with ASD and patients with PD function differently on aspects of social cognition? If this question results in a confirmation, the measurement of social cognition with chosen measures (see below) can validly differentiate in the phenomenological overlap between ASD and PD, contributing to the earlier mentioned clinical practice dilemma. An ASD and PD group will be compared on both self-report and ability measures of social cognition (aspects like Theory of Mind, emotion recognition and ability to regulate emotions). The Emotional Quotient-inventory (EQ-i), the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), the Bermond-Vorst Alexithymia Questionnaire (BVAQ) and the Strange Stories Task (SST) are used to assess the various aspects of the social cognition process. For part of the data, the groups are also compared with data from non-patients.

Given the described social cognitive problems in ASD, impairments can be expected on the ability to read intentions and emotions (e.g., Atkinson 2009, Baron-Cohen et al. 1997, Baron-Cohen and Wheelwright 2004, Domes et al. 2008, Grossman et al. 2000, review of Hill and Frith 2003, Kanner 1943, Konstantareas and Steward 2006). For the PD group, because of their negativity bias in perceiving self and others, patients can be expected to estimate themselves as more impaired on reading emotions and intrapersonal functioning. Patients with PD are expected to show more impairment on the emotionalising part of social cognition (e.g., Brown and Cohen 2010, Domes et al. 2009, Lynch et al. 2006). Aspects of social cognition can be measured by self-report and ability measures. Patients with ASD have been shown to be able to give a realistic estimation

of own functioning on self-report scales, as compared to estimations by their family context; that is, no contra-indications seem to be present for using self-report measures in this group (Ozonoff et al. 2005).

In the stating of hypotheses, a difference is made between these self-report and ability measures. Put shortly, with respect to ability, we hypothesize that: (a) Patients with ASD perform lower (more impaired) on the ability to read intentions and emotions, than patients with PD. With respect to self-report, we hypothesize that: (b) Patients with PD estimate themselves as more impaired on the ability to read emotions and intrapersonal functioning, than patients with ASD. With respect to both self-report and ability, we hypothesize that: (c) Both patients with ASD and patients with PD estimate themselves as more impaired on aspects of social cognition, and actually have a lower ability on these aspects, than non-patients (control data). For the PD patients, the accent of impairments is expected on the emotionalising part of social cognition. Within the ASD and the PD groups, differences between diagnostic subgroups will be studied in an explorative style because of smaller n sizes (lower power).

Method

Participants

Participants consisted of 51 patients diagnosed with an ASD (ASD group) and 68 patients diagnosed with a PD (PD group). The ASD group consisted of 46 males and 5 females in the age of 19 to 62 years ($M=35.04$, $SD=13.5$). The PD group consisted of 22 males and 46 females in the age of 15 to 65 years ($M=35.88$, $SD=13.7$). Evaluated by Lezak's model of normal distribution, both groups had an average Intelligence Quotient (IQ) (ASD group $M=93.8$, $SD=8.3$; PD group $M=93.9$, $SD=13.0$); the inclusion threshold for IQ was set on ≥ 70 . For the ASD group, the diagnoses were: 2 autistic disorder, 18 Asperger's disorder and 31 Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). For the PD group, the cluster of the primary personality disorder diagnosis was determined (also for the Not Otherwise Specified group): 1 cluster A, 35 cluster B, 32 cluster C. All patients were treated at the Vincent van Gogh institute for mental health care. Non-patient data for EQ-i and MSCEIT tests were derived from PEN Psycho-diagnostics (Bögels 2010) and matched with both the ASD and PD group, for age and sex (no IQ data present). For the ASD group, non-patient controls for EQ-i ratings were 47 males and 5 females, mean age 35.65 ($SD 8.91$). For the MSCEIT ratings non-patient controls for the ASD group were 47 males and 5 females, mean age 35.54 ($SD 11.52$). For the PD group,

non-patients control data for the EQ-i were derived from 20 males and 34 females, mean age 36.24 (SD 9.90). For the MSCEIT non-patient control data for the PD group were scores from 22 males and 43 females, mean age 36.20 (SD 11.98).

Materials

Social cognition measures consisted of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT; Mayer et al. 2003), the Emotional Quotient Inventory (EQ-i; Bar-on 1997), the Strange Stories Task (SST; Happe 1994) and the Bermond-Vorst Alexithymia Questionnaire (BVAQ; Bermond and Vorst, 1996). An IQ test was performed as a check and for the use as covariate (see procedure).

Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT)

The MSCEIT is an ability measure of social cognition, involving emotional processing. It assesses branches: emotion perception (ability to recognise how one and others feel; task with pictures of facial expressions), understanding (knowledge and reasoning about complex emotions and the way emotions unfold), facilitation (ability to generate and reason with emotion) and regulation of emotion (management of emotion in oneself and others).

These branch descriptions clearly show overlap with aspects of the earlier mentioned social cognition definition (integrated processing of socio-emotional information needed to direct interaction; Swaab et al. 2011). The MSCEIT consists of 141 items across 8 tasks; two tasks comprise each of the 4 mentioned branch scores in the Mayer and Salovey 4-factor model of emotional intelligence. Two area scores are derived from the model; the experiential ability (being able to perceive, experience and reason with emotionally laden information) and the strategic ability (being able to understand emotionally laden information and manage emotions). Total, subscale and individual items are scored through a web-based scoring program (Multi-Health Systems, Inc, Canada), using unadjusted consensus norms from a large normative sample, where participants receive a score based on the proportion of individuals in the normative sample that endorse their response to a particular question. Total and branch scores are automatically calculated and scaled with a mean of 100 (SD=15), with higher scores reflecting better emotional intelligence (Eack et al. 2010, Mayer et al. 2003). In psychometric properties studies which were described by Brackatt and Salovey in 2006, the MSCEIT showed a full-test split-half reliability of .93 for consensus scoring (which is used for the present study), area scores reliabilities were .90 and .90 and branch score reliabilities were between .76 and .91. Test-retest reliability of the full-test MSCEIT over a three-week interval was $r(59) = .86$ in a college student sample. Satisfying factor-structure is present and the MSCEIT showed content and structural validity as well as discriminant, convergent, predictive, and incremental validity. Sex and age corrections were applied for the ASD and PD group comparisons (primarily to compensate for the sex differences between the groups). For the matched control data, only raw composite scores were present; they were compared with those for the ASD and PD groups.

The Emotional Quotient Inventory (EQ-i)

The EQ-i is a self-report measure of social cognition aspects like emotional intelligence. It consists of 133 items, with a response rating scale (5-point), ranging from "very seldom true or not true of me" to "very often

true or true of me". Scores are provided and adjusted to standard scores for total EQ, 5 EQ composite scales, 15 EQ content scales and 4 Validity indicators (Bar-on 1997). The mean standard score is 100 (SD=15) (Dawda and Hart 2000). The 5 EQ composite scales include Intrapersonal functioning (content scales emotional self-awareness, assertiveness, self-regard, self-actualization, independence), Interpersonal functioning (content scales empathy, social responsibility, interpersonal relationships), Adaptation (content scales reality testing, flexibility, problem solving), Stress management (content scales stress tolerance, impulse-control), and General mood aspects (content scales optimism, happiness). Higher scores generally indicate a self-estimate of more adequate functioning, although scores high above average can be inadequate as well (for example, over social responsible behaviour). Dutch norms were provided by the Catholic University Brabant (Nederlands Instituut voor Psychologen NIP 2005). Test-retest reliabilities of .73 and .78 were mentioned by Brackett and Mayer (2003). Bar-on described positive validity results (2005).

Strange stories task (SST)

The SST (Happe 1994, Spek et al. 2010) consists of short stories. It assesses participant's ability to attribute mental states to others, by stories featuring a pretend event, a joke, a lie, a white lie, a figure of speech, and bluffing. After the participant's basic understanding of the story is assessed, an open-ended question is presented to assess the participant's understanding of the character's mental state. The score is derived from the total number of physical instead of mental state responses and the total number of wrong responses in understanding the stories. The SST has positive reliability and validity results on measuring Theory of Mind as part of social cognition in children (Kaland et al. 2002). A non-patient group made very few faults on the SST; $n = 32$, M failure score = .69, SD 1.09 (Spek et al. 2010). The strange stories task that was used in the present study, was translated by forward-backward procedure in Dutch by Spek et al. (2010). It contains eight stories, derived from Happe's original task (1994) as being the most difficult for adults. Stories were scored by a second rater, and the degree of concordance was 97 % for the 'correct answer score' and 95 % for the 'mental explanation score'.

Bermond-Vorst Alexithymia Questionnaire (BVAQ)

The BVAQ (Bermond and Vorst 2001) is a 40-item self-report measure, which comprises two parallel versions each of 20 items. Each item is rated on a five-point Likert scale, ranging from 1 (strongly agree) to 5 (strongly disagree). The five factors indicating the recognition and regulation of one's own emotions (alexithymia is the deficit in doing this) are: verbalizing (self-reported ability to describe and communicate own emotional states), fantasizing (self-reported ability to fantasize, imagine, day dream), identifying (self-reported ability to define reasons for being aroused), emotionalizing (self-reported ability to experience emotional arousal with emotion triggering events) and analysing (self-reported tendency to seek out own emotional reactions (Bermond and Vorst 2001). For each factor, high scores are indicative of alexithymia. Psychometric properties of the BVAQ have shown to be sufficient; reliability ranges from .67 to .87 for subscales and validity was in an acceptable range (Müller et al. 2004, Vorst and Bermond, 2001).

In a study of Berthoz and Hill (2005) on ASD and alexithymia, patients were able to reflect on their emotions in an emotionalizing and fantasizing way, but less in a cognitive way (verbalizing, identifying and analyzing).

Procedure

Participants were selected by administrative searches on diagnosis (ASD or PD) with as few as possible comorbid diagnoses (for example, diagnoses like schizophrenia and major affective disorders were excluded). To complete the described tasks, participants required two hours. Diagnoses of participants (ASD or PD) were checked, using the Longitudinal Expert Evaluation using All Data (Lead-method; Spitzer 1983): an expert diagnosed the patient by using information from multiple sources (patient, family, team as stated in patient file). The SCID-I and II screening questionnaires were also conducted for this purpose (Groenestijn et al. 1999, Weertman et al. 2000). For ASD, during the Lead-method, the presence of (hetero-) anamnestic information (focusing on ASD) was checked. As a check and for the use as covariate, an IQ screening test, the Dutch version of the National Reading Test for Adults (NLV) was used; the threshold was put at 70.

Statistical analyses

A comparative design with exploratory aspects was used for this study. To determine the domains of the instrument's major subscales used in this study, a principal component analysis with oblique rotation was used, with the number of factors extracted, based on the scree plot. In order to contain relevancy and power of analyses, the major scales of test measures (13) were taken into the principal component analysis: the verbalising, fantasising, emotionalising, analysing and identifying scales of the BVAQ, the intrapersonal, interpersonal, stress management, adaptation and general mood scales of the EQ-i, the experiential and strategic area scales of the MSCEIT and the total failure score of the SST. After data-reduction, (M)ANCOVA's were conducted for variables that met basic assumptions (normality, homogeneity of variances, linearity). Ordinal regression (PLUM) analysis was conducted for factor-scales (part of factor domains A, B and C), not meeting these basic assumptions; the advantage of this analysis is that it enables accounting for covariates, contrary to Mann-Whitney tests. The independent variable for most hypotheses was diagnosis (ASD, PD, non-patients). The dependent variables were the scores on specific subscales. Sex, age and IQ were used as covariates (IQ not for comparisons to non-patients, as we had no IQ estimates for them). Means were adjusted to covariates. SPSS version 18.0 was used. Missing values were present for 21 patients on part of the BVAQ due to a lacking page of items. For 1 patient the EQ-i was not present and for 1 patient the MSCEIT computer system failed for part of the scoring. Cases with missing values were excluded on a pairwise basis, in order to be able to still analyse the remaining case data on other measures. As 34 statistical tests were done, the p-level was adjusted using a False Discovery Rate adjusted p-level of 0.01214 (Narum 2006).

Results

After doing a Principal Component Analysis with oblique rotation (see method for variables taken into account), four "social cognition" factors were derived and the following categorization in factor scales was made. A) Ability to read and regulate intentions and emotions, linking with hypothesis a, focusing on ability; the task scales "experiential ability", "strategic ability

(including management of emotions)" (both MSCEIT) and the "total failures SST" loaded highest, the latter in negative direction. The explained variance was 13.06 %. B) Self-estimate of ability to read emotions, linking with hypothesis b, focusing on self-estimate; the scale "interpersonal functioning" (EQ-i) and the scales of verbalising, identifying, emotionalising and analysing of emotions (BVAQ) loaded highest, only interpersonal functioning in positive direction. The explained variance was 18.69%. C) Self-estimate of intrapersonal functioning, also linking with hypothesis b, focusing on self-estimate; "intrapersonal functioning", "stress management", "adaptation" and "mood" (EQ-i) loaded highest, all in positive direction. The explained variance was 28.72 %. D) Self-estimate of ability to fantasize; scale "fantasizing" (BVAQ) loading highest, in negative direction. The explained variance was 8.01 %. Factor scale D was not taken into account for analyses due to the mentioned lower n for the BVAQ measure. Hypothesis c, concerning both ability and self-report comparisons of ASD and PD groups with controls, links to both domain A, B and C.

(Multivariate) analyses of covariance (MANCOVA's) were conducted to compare the ASD, PD group and controls (hypothesis c). MANCOVA's were only conducted with the factor variables (variables loading on A, B or C), for which basic assumptions of normality, homogeneity of variances, and linearity were met. If not, an ordinal regression (PLUM) was conducted. In all analyses, diagnosis was used as the between-subject factor. In addition, IQ, sex and age were used as covariates for the ASD and PD group comparisons (for controls, no IQ data present). Results that are not mentioned were non-significant. All Box M's tests for equality of covariance matrices were non-significant. As mentioned, due to the 34 statistical tests that were done, the p-level was adjusted using a False Discovery Rate adjusted p-level of 0.01214 (Narum 2006).

Ordinal regression and (M)ANCOVA analyses were conducted for both the ASD and the PD group with matched controls (sex, age) on the EQ-i and MSCEIT, linking with hypothesis c. Scores, available for analysis, were the raw composite scale scores for the EQ-i and raw branch scores for the MSCEIT. Results showed that both the ASD and PD group are impaired on the strategic regulation of emotions, as compared to controls. In addition, both the ASD and PD group report worse functioning on ability to read emotions and intrapersonal functioning (EQ-i major scales intra, and interpersonal functioning, stress management, adaptation and general mood). See **tables 1, 2 and 3** for descriptive statistics, group and covariate effects.

The univariate analysis for the comparison between ASD and PD on reading and regulating intentions and emotions (domain A, linking with hypothesis a, concerning ability comparison), with MSCEIT experiential area as dependent variable, showed no significant main effect for diagnosis, sex or age, but did reveal a main effect for IQ (higher IQ performing better). See **table 4** for descriptive statistics and **table 5** for ANCOVA.

The multivariate analysis, with (domain B, linking with hypothesis b, self-estimate comparison) self-estimate of ability to read emotions-scales as dependent variables, showed (only) a significant main effect for diagnosis; the significant diagnosis effect was specifically visible on the interpersonal functioning-scale: the ASD group reporting worse functioning than the PD group. See **table 4** for descriptive statistics and **table 5** for MANCOVA. The interpersonal functioning-scale is, as mentioned, a major EQ-i scale; when analysing the

Table 1. Descriptive statistics variables, for the comparison of ASD and PD groups with controls on major scales EQ-i and MSCEIT

Dependent variables	ASD		Controls		PD		Controls	
	Median	Quartiles	Median	Quartiles	Median	Quartiles	Median	Quartiles
EQ-i major scales:								
Intrapersonal functioning	122.50	108.00-139.75	163.00	151.25-171.00	104.50	89.25-115.50	158.00	141.50-173.5
Interpersonal functioning	80.00	70.50-85.25	98.00	86.00-107.75	87.00	80.00-94.00	101.50	94.00-108.00
Adaptation	82.00	76.00-89.00	96.00	89.50-102.75	76.00	71.00-84.00	97.00	88.75-104.25
Stress management	57.00	51.25-66.00	67.00	62.00-73.00	52.00	43.00-58.25	64.50	57.75-71.25
General mood	58.00	46.25-64.75	72.00	65.25-77.00	47.00	40.00-58.00	71.50	65.00-77.00
MSCEIT major area scales:								
Emotional experiential	.48	.41-.53	.48	.40-.53	.47	.41-.52	.49	.41-.53
Emotional strategic	.43	.39-.47	.46	.42-.48	.43	.39-.46	.47	.45-.50

Note:

-For the controls data, available for analysis were the raw composite scale scores for the EQ-i and the raw branch scores for the MSCEIT.

-In this table, for both normally and not-normally distributed variables the medians and quartiles are stated.

Table 2. Ordinal regression analysis with major scales of the EQ-i and MSCEIT, for the ASD comparison with controls

Dependent variables	Diagnosis (ASD)			Covariate sex			Covariate age			df=1
	Estimate	Wald	p	Est	Wald	p	Est	Wald	p	
EQ-i major scales:										
Intrapersonal functioning	-3.29	49.75	<.001*	-.48	.65	.42	.01	.54	.46	
Interpersonal functioning	-2.55	38.02	<.001*	1.28	4.54	.03	.02	2.55	.11	
Adaptation	-2.62	39.36	<.001*	-.90	2.27	.13	.02	2.26	.13	
Stress management	-1.86	24.03	<.001*	-1.46	5.81	.02	.01	.86	.35	
General mood	-2.65	39.75	<.001*	-.10	.03	.87	.02	2.57	.11	
MSCEIT major area scales:										
Emotional experiential	.08	.06	.81	.58	.96	.33	.01	.17	.68	
Emotional strategic	-.86	6.16	.01*	.56	.89	.35	.002	.02	.89	

Note: -* = Significant result; False Discovery Rate BY method, $k = 34$, $p < .012$. Es = effect size partial eta squared (due to the 34 statistical tests that were done, the p-level was adjusted using a False Discovery Rate adjusted p-level of 0.01214 (Narum, 2006))

-For normally distributed variables, (M)ANOVA's were conducted, leading to the same (significant) results

Table 3. Ordinal regression analysis with major scales of the EQ-i and MSCEIT, for the PD comparison with controls

Dependent variables	Diagnosis (PD)			Covariate sex			Covariate age			df=1
	Estimate	Wald	p	Est	Wald	p	Est	Wald	p	
EQ-i major scales:										
Intrapersonal functioning	-4.47	68.11	<.001*	-1.19	8.89	<.01*	.03	2.52	.11	
Interpersonal functioning	-2.27	34.82	<.001*	.11	.08	.78	.01	.19	.66	
Adaptation	-3.49	60.73	<.001*	-1.74	18.08	<.001*	.02	.93	.34	
Stress management	-2.87	48.82	<.001*	-1.92	21.09	<.001*	.01	.08	.77	
General mood	-4.09	65.86	<.001*	-.78	4.01	.05	.03	4.14	.04	
MSCEIT major area scales:										
Emotional experiential	-.22	.52	.47	.13	.13	.71	-.01	.52	.47	
Emotional strategic	-1.43	19.28	<.001*	-.01	.001	.98	-.01	.63	.43	

Note:

-* = Significant result; False Discovery Rate BY method, $k = 34$, $p < .012$. Es = effect size partial eta squared (due to the 34 statistical tests that were done, the p-level was adjusted using a False Discovery Rate adjusted p-level of 0.01214 (Narum, 2006))

- For normally distributed variables, (M)ANOVA's were conducted, leading to the same (significant) results

Table 4. Descriptive statistics & analyses normally distributed variables, part of factors A, B and C, means adjusted for covariates age, IQ and sex

Dependent variables	Autism Spectrum Disorder		Personality disorder		p (df=1)
	Mean	SD	Mean	SD	
A) Ability to read and regulate emotions: Emotional Experiential (MSCEIT)	96.81	14.78	92.22	13.79	.16
B) self-estimate of ability to read and regulate emotions:					
Interpersonal (EQ-i)	77.35	19.49	94.19	17.39	.001*
Identifying em. (BVAQ)	22.00	7.13	23.37	7.94	.50
C) self-estimate of intrapersonal functioning:					
Intrapersonal functioning	75.37	15.72	67.30	18.87	.05
Adaptation (both EQ-i)	74.34	16.79	77.48	16.89	.41

Notes:

-*= significant result on base of False Discovery Rate, BY method, $k=34$, $p < .012$ (Due to the 34 statistical tests that were done, the p-level was adjusted using a False Discovery Rate adjusted p-level of 0.01214 (Narum, 2006)).

- For factor A, an ANOVA analysis was conducted; for factors B and C MANOVA's were conducted.

Table 5. Analyses on normally distributed variables, part of domains A, B and C

Dependent variables	Diagnosis (ASD and PD)			Covariate sex			Covariate age			Covariate IQ		
	F	p	es	F	p	es	F	p	es	F	p	es
A) Ability to read and regulate emotions: Emotional Experiential (MSCEIT)	(1,109) = 2.00	.16	.02	.16	.69	.001	5.97	.02	.05	9.83	<.01*	.08
											Higher IQ better functioning	
B) self-estimate of ability to read and regulate emotions:	(2,88) = 9.4	<.001*	.18	.82	.45	.02	2.72	.07	.06	1.11	.33	.03
Interpersonal (EQ-i)	(1,89) = 12.30	.001*	.12	1.43	.24	.02	4.55	.04	.05	.51	.48	.01
	ASD report worse functioning than PD											
Identifying em. (BVAQ)	(1,89) = .45	.50	.01	.02	.90	.00	3.42	.07	.04	.71	.40	.01
C) self-estimate of intrapersonal functioning:	(2,108) = 5.89	<.01*	.10	3.73	.03	.07	3.81	.03	.07	2.80	.07	.05
	ASD report better intra funct. than PD											
Intrapersonal functioning	(1,109) = 3.91	.05	.04	.49	.49	.004	1.23	.27	.01	4.93	.03	.04
Adaptation (both EQ-i)	(1,109) = .693	.41	.01	6.06	.02	.05	7.00	<.01*	.06	4.41	.04	.04

Notes:

-* = Significant result; False Discovery Rate BY method, $k = 34$, $p < .012$. Es = effect size partial eta squared (due to the 34 statistical tests that were done, the p-level was adjusted using a False Discovery Rate adjusted p-level of 0.01214 (Narum, 2006))

-For factor A, an ANOVA analysis was conducted; for factors B and C MANOVA's were conducted

significant effect further by looking at the content of this major scale, the empathy and interrelationships fit with this effect ($F(1,87) = 16.2$, $p < .001$ and $F(1,87) = 5.2$, $p < 0.3$); the ASD group reporting worse functioning than the PD group.

The multivariate analysis, with (domain C, also linking with hypothesis b, self-estimate comparison) self-estimate of intrapersonal functioning-scales as dependent variables, showed significant main effects for diagnosis and age, but not for sex and IQ. Patients with ASD report better functioning than patients with PD, on intrapersonal functioning. A significant effect was found on the adaptation scale for age (lower age reporting better functioning). See **table 4** for descriptive statistics and **table 5** for MANCOVA. The intrapersonal functioning is, as mentioned, part of the EQ-i; when analysing the significant effect further by looking at the subscales, the self-actualisation scale fits with this effect ($F(1,109) = 8.7$, $p < .01$), patients with ASD reporting better functioning.

Ordinal regression (PLUM) analyses were conducted for scales not meeting basic assumptions. For the descriptive statistics and results of this analysis see **table 6** and **7**. In domain A (linking with hypothesis a, ability comparison), ability to read and regulate intentions and emotions, significant effects were found for IQ and age on the strategic regulation of emotions, but not for diagnosis and sex. In domain B, self-estimate of ability to read emotions, no significant effects were present for diagnosis, age, IQ or sex. In domain C, self-estimate of intrapersonal functioning, the only significant effect was present for age on stress management; higher age related to report of lower management.

MANCOVA's for scales meeting basic assumptions (part of domains A, B and C) and ordinal regression for scales not meeting basic assumptions, did yield no within group differences (differences between various ASD-diagnoses, and differences between various PD-clusters). Due to the low number, cluster A patients ($n=1$), were excluded.

Table 6. Descriptive statistics not normally distributed variables, part of factors A, B and C

Dependent variables	Autism Spectrum Disorder		Personality disorder	
	Median	Quartiles	Median	Quartiles
A) Ability to read and regulate emotions: Emotional Strategic (MSCEIT)	87.48	82.61-94.96	85.98	80.46-93.22
Failure score (SST)	1.00	0.00-5.00	1.00	0.00-2.00
B) self-estimate of ability to read and regulate emotions: Verbalising (BVAQ)	31.00	23.50-35.00	28.00	19.00-34.75
Emotionalising (BVAQ)	22.00	16.75-27.00	16.50	13.00-19.75
Analysing (BVAQ)	20.50	13.75-27.00	17.00	12.00-23.00
C) self-estimate of intrapersonal functioning: Stress management (EQI)	87.00	76.00-98.00	85.00	70.00-93.00
Mood (EQ-i)	77.00	66.00-93.00	66.00	58.00-85.00

Table 7. Ordinal regression analysis with variables (part of factors A, B and C, not normally distributed), PLUM

Dependent variables	Diagnosis (ASD and PD)			Covariate sex			Covariate age			Covariate IQ			df=1
	Estimate	Wald	p	Est	Wald	p	Est	Wald	p	Est	Wald	p	
A) Ability to read and regulate emotions: Emotional Strategic (MSCEIT)	-.23	.32	.57	-.91	4.02	.05	-.10	41.25	< .001*	.07	17.23	< .001*	
Failure score (SST)	.93	4.45	.04	.40	.70	.40	.03	3.29	.07	-.04	5.85	.02	
B) self-estimate of ability to read and regulate emotions: Verbalising (BVAQ)	.94	3.97	.05	.84	2.81	.09	.02	2.50	.11	.002	.02	.89	
Emotionalising (BVAQ)	.99	4.38	.04	-.105	4.31	.04	.01	.96	.33	.007	.14	.71	
Analysing (BVAQ)	.59	1.60	.21	-.27	.29	.59	.03	3.28	.07	-.01	.10	.76	
C) self-estimate of intrapersonal functioning: Stress management (EQ-i)	.13	.09	.76	-.60	1.75	.19	-.05	11.46	.001*	.03	3.24	.07	
Mood (EQ-i)	.59	2.06	.15	-.24	.28	.60	-.01	.11	.74	.02	.89	.35	

Note:

-* = Significant result; False Discovery Rate BY method, $k = 34$, $p < .012$. Es = effect size partial eta squared (due to the 34 statistical tests that were done, the p-level was adjusted using a False Discovery Rate adjusted p-level of 0.01214 (Narum, 2006))

Discussion

The present study is one of the first that aims to contribute to differential diagnosis of Autism Spectrum Disorders and Personality Disorders (disorders having phenomenological overlap), by measuring social cognition. Results show that patients with ASD estimate themselves as more impaired than patients with PD on reading emotions, whereas patients with PD report themselves as more impaired on intrapersonal functioning. Patients with ASD and patients with PD do not differ in their ability to read and regulate emotions and both groups report more impairment on aspects of social cognition on a self-report measure and on a part of an ability measure, as compared to non-patient data. Furthermore, ASD and PD subtyping (Autistic disorder, Asperger's disorder, PDD-NOS, PD clusters A, B and C) does not lead to distinguishable findings. This latter finding fits the subdividing discussion as mentioned in other studies (e.g. for ASD: Witwer and Lecavalier 2008, for PD: Westen and Shedler 1999).

The results implicate the following with respect to

the stated hypotheses.

As to the negativity bias hypothesis (Domes et al. 2009, Lynch et al. 2006), this was confirmed for the intrapersonal part, patients with PD reporting their intrapersonal functioning as more impaired. This result fits the earlier mentioned PD sensitivity for a negative cognitive bias in (primarily) the perception of self. Consequently, when one is tended to reflect negatively on own functioning, one may feel less control to participate in daily life, which can also lower one's well being (Lefcourt 1982, Seifer et al. 1992). In accordance with this, Arntz et al. (2011) describe a tendency in Borderline, Avoidant and Dependent PD patients to criticize themselves and have negative emotional responses combined with low solution-focused strategies, influencing emotion regulation as well (also mentioned in e.g., Dijke et al. 2010, Johnson et al. 2003). Patients with ASD indicate themselves as impaired in interpersonal functioning, but their overall well-being does not get influenced so thoroughly as in PD. Explanatory, patients with ASD may be less emotionalising, but more cognitively reflective on

own functioning than patients with PD. Consequently, the confrontation with functional impairments may lead to a lesser amount of “emotional insight and/or suffering” in ASD. Nevertheless, the negativity bias in PD is just one possible explanation for their scores on these measures. It does call for further research on this part, in addition to a deeper view on the differentiation between emotionalising and cognitive parts of emotion perception, regulation and its influence on patient responses.

The social cognitive ability hypothesis, expecting patients with ASD to function more impaired than patients with PD on reading and regulating emotions, was not confirmed. The finding that there are no differences between ASD and PD on social cognitive ability measures also provides a limitation for our study; one could say that both patients with ASD and PD have problems in reading and regulating emotions, but one still cannot say if these problems are similar in its specific nature and causes. That is, focusing on the social cognitive ability function as operationalized by the selected tasks we used, may not sensitively have measured detailed subdivisions in social cognitive abilities and/or specific key impairments. Furthermore, within the stated hypothesis, no differentiation was made between emotionalising and more cognitive aspects of social cognition. Consequently, when aiming to focus on a broad construct like social cognition to differentiate between disorders, awareness concerning the sensitivity of selected tasks to pick up detailed differences between groups is important and needs more research. It also suggests that findings need to be embedded within a more detailed, tailored profile of strengths and weaknesses, that forms an (dimensional) overview of psychopathology and primarily gives information concerning the specific accents and intensity of impairments. Furthermore, bio-physiological measures like EEG/MRI measures would give an even deeper and detailed look under the behavioural level than the tasks we performed in the present study; electrophysiology is thereby an important technique for future research.

The hypothesis that patients with ASD and PD estimate themselves as more impaired on social cognition and actually function worse on part of an ability measure than controls is confirmed. For both ASD and PD, keeping in mind earlier literature findings from the introduction concerning interpersonal, intrapersonal functioning and emotion regulation this fits the expectations. The knowledge that patients with PD show problems on both, though not all self-report and ability measurements, sheds an additional light on the earlier mentioned negativity bias hypothesis. Patients with PD have a negative estimate of own social cognitive functioning, but they actually do have impairments in emotion regulation. One could state that, these results indicate that patients with PD have a realistic view of own vulnerabilities instead of a negativity bias. Nevertheless, these results do not rule out the negativity bias either; they could fit the interplay between (social) cognition and emotion in the same model, as described earlier by Izard (2009). In other words, having negative cognitions can influence one's functioning on -social cognitive- emotional ability measures, but the functioning on emotionally laden measures (as a stimulus) in turn can influence the negative cognitions as well. This implicates self-report and ability measures can give insight in the nature and intensity of impairments in ASD and PD, but it definitely does not give (and is not meant to give) a linear way of explanatory understanding.

As mentioned before, the diagnostic knowledge that both the ASD and PD group are vulnerable in social cognition has therapeutic implications as well. While ASD is generally seen as a chronic disorder and therapeutic interventions are mostly focused on care (e.g. Vermeulen 2005), PD is currently seen as relatively more open to change, and therapies are successfully focused on cure, striving for healthier ways of regulating emotions and social interactions (e.g. Young et al. 2003). It would be challenging to speculate about ASD as having changeable social cognitive aspects as well; studies involving emotion recognition and Theory of Mind training are promising (e.g. Ozonoff and Miller 1995, Williams et al. 2012, Vissers and Honée-van Zijll de Jong in preparation).

With regard to limitations of our study; looking back, it might have been contributing to include a hetero-anamnestic (developmental) interview in the comparison between ASD and PD. Whereas this measure is custom for ASD diagnostic practice, it is not for PD diagnostic practice. In future research, social cognition aspects as present within the biographical information, could also have contributing worth in differentiating between ASD and PD. Furthermore, although all patient files were checked by Lead-method, a more thorough overview of the detailed content and quality of diagnostic measures that were undertaken in the patient groups (prior to this study) would have been more precise. Perhaps, comparing various PD's could have shown differences in performance on social cognition, but due to lesser n sizes per PD we chose to compare clusters (see method). Nevertheless, firstly, the n sizes do, given an official administrative search, show a realistic representation of cluster diagnoses in our clinical practice. Secondly, in line with the dimensional future of DSM, estimating degrees of impairment may have more worth, than subdividing disorders. With regard to types of tests (ability measurement by MSCEIT versus self-report measurement by EQ-i), it shows that ability and self-report measures lead to different results. In other words: a negative self-reporting tendency does not automatically relate to worse functioning on actual capacities. These findings are supported by several other studies, and it makes the evaluation of which kind of tests to use important, depending on study aims (Beblo et al 2010, Hertel et al. 2009, Leible and Snell 2004, Peter et al. 2013). Against the background of the emotionalising reflection impairment-hypothesis, one could be tended to state that self-report measures are less suitable for patients with ASD. Contrary, as earlier mentioned, patients with ASD are shown to be able to give a realistic estimation of own functioning on self-report scales, as compared to estimations by their family context (Ozonoff et al. 2005). Further research is needed to test this statement. Furthermore, one could state that the EQ-i and MSCEIT do not specifically measure social cognition, but rather measure aspects like emotion (regulation), that is linked to the social cognition process. Nevertheless, the amount of overlap between these constructs is not sufficiently clear yet, as a consequence of which conclusions should be taken cautiously. Both patient groups do show impaired functioning on the emotion regulation ability MSCEIT scale, as compared to non-patient data. This construct was not thoroughly measured in the present study, but, investigating the (socio-emotional) negativity bias in patients with PD, could lead to greater insight in (differentiation between) the neuropsychological profiles in ASD and PD. In future research experientially based emotion regulation tasks should be developed and performed (p. e. Emotion Induction tasks).

In addition to the earlier mentioned limitations of our study, we also realise that the present effect sizes are fairly small. Furthermore, the missing values on certain measures (BVAQ, see method), made our *n* sizes fluctuate over analyses, which interferes with the power of statistical tests. In addition, the lack of IQ data in the ASD-controls and PD-controls comparison is a limitation, mostly because IQ may have influenced the differences found in these comparisons and it could have been taken into account as a covariate. Lastly, in future research we'll strive for the collection of own control data, for all the measures conducted. Concluding, neurocognition contributes to insight into the phenomenology of disorders by putting a deeper look, in addition to the topographical behaviour viewpoint. That is, by mapping part of the relation between brain and behaviour. As mentioned, the use of electrophysiology is a recommendation for future research, in order to gain an even deeper detailed look under the surface. Nevertheless, measuring neurocognitive functioning seems highly relevant, provided that it is embedded within a detailed and primarily dimensional profiling of strengths and weaknesses, combining all three levels of brain, cognition and behaviour. Measuring social cognition as performed in the present study, does give insight in functioning of patients with ASD and patients with PD. However, it needs embedding within more detailed profiling. Furthermore, hetero-anamnestic information concerning development throughout the life-span is needed. In other words, social cognition needs to be measured along with these other diagnostic steps, to be able to contribute to differentiating the disorders. For social cognition, it seems particularly useful to look at self-report measures. These can shed light on one's participation in social life tasks and quality of life, thereby touching possible treatment strategies in a tailored way. For example, focusing on self-actualization behaviour and a more healthy strategy in emotionalising may improve the intrapersonal functioning of patients with PD, while focusing on empathic perspective taking (for example, by deictic framing training, ToM training as well as emotion recognition training (e.g., Janssen et al. 2014, Ozonoff and Miller 1995, Williams et al. 2012, Vissers and Honée-van Zijl de Jong in preparation) may improve interpersonal functioning of patients with ASD. These diagnostic and treatment strategies can in turn lead to insight and understanding in patients, which can influence their in-control thoughts, needed for a more healthy quality of life. Furthermore, focusing on a detailed neurocognitive strengths and weaknesses profile seems to transcend the relevance of subdividing disorders like several ASD's and PD's. In order to form this detailed profile, the tasks as used within the present study can be of help to a clinician, concerning the aspect of social cognitive functioning. Furthermore, patient's own estimates of their functioning and suffering areas as measured by the EQ-i can provide therapeutic entrances. Nevertheless, current findings do call for further research, to explore further whether ASD and PD differences are indeed associated with accents in emotionalising versus cognitive reflection and abilities, a negativity bias, and/or (out of) control-thoughts.

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